Photodisruptive Neodymium:Yttrium-aluminum-garnet Laser in the Management of Premacular Subhyaloid Haemorrhage

H Durukan,[1] Y Akar,[2] G Sobacı,[1] S Karagül,[1] Z Bayraktar[1]
[1] Department of Ophthalmology, Gülhane Military Medical Academy, Ankara
[2] Department of Ophthalmology, Akdeniz University, Antalya, Turkey

This report is of a patient with acute idiopathic premacular subhyaloid haemorrhage, which was treated successfully by subsequent Q-switched neodymium:yttrium-aluminum-garnet laser hyaloidotomy via the transcorneal route.

Key Words: Lasers, Macula lutea, Haemorrhage, Visual acuity

Asian Journal of Ophthalmology 2002;4(4):10-12.

Introduction

A 21-year-old, otherwise healthy, man was referred to the clinic with sudden visual loss to counting fingers at 20 cm in the right eye for 20 days' duration. There was no history of systemic or ocular disorders, trauma, or surgery. No further identifiable cause for subhyaloid haemorrhage was found upon systemic evaluation. Complete ophthalmological examination of the left eye was normal with a visual acuity of 20/20.

Anterior segment examination of the right eye was normal. Funduscopy of the right eye revealed a round, well circumscribed, dome-shaped haemorrhage with a convex surface overlying the posterior pole, extending between the temporal vascular arcades, consistent with a subhyaloid or subinternal limiting membrane (subILM) haemorrhage (Figure 1). Colour fundus photographs were obtained before and after the haemorrhage was treated. The size of the pretreated haemorrhage was estimated to be 5 disc diameters.

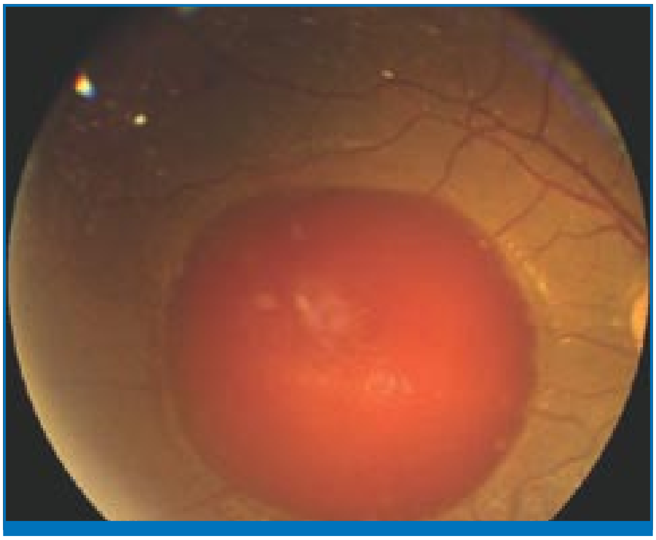
Q-switched neodymium:yttrium-aluminum-garnet (Nd:YAG) laser was performed on the posterior hyaloidotomy of the right eye over the dark, brown haemorrhage via the transcorneal route. Full pupillary dilatation was achieved with cyclopentolate 1% and phenylephrine 10%. Using simple contact anaesthesia with proparacaine, a Goldmann 3-mirror fundus contact lens was used to allow focussing of the Nd:YAG laser aiming beam. The aiming beam was precisely focussed on the surface of the posterior hyaloid membrane at the inferior edge of the subhyaloid haemorrhage to facilitate gravity-induced drainage.

The starting energy was set at a pulse power of 1.3 mJ (50 Hz burst mode, 10° cone angle, and a single pulse mode). The energy was progressively increased until a clear rupture of the target tissue was obtained. The rupture of the posterior hyaloid membrane, at a location distant from the fovea and retinal blood vessels but with a sufficient thickness of blood to protect the underlying retina, was achieved with a power of 8.7 mJ. Fifteen low-energy bursts of Nd: YAG laser were applied to perforate the anterior surface of the haemorrhage. The total energy required was 155.3 mJ.

At the end of the procedure, the haemorrhage instantaneously drained into the vitreous cavity (Figure 2), resulting in a fast visual recovery. The following day, visual acuity was 5/20, which progressively improved each day, achieving a final visual acuity of 20/20 by post-laser day 7. Fluorescein angiography, performed at post-laser day 4, did not demonstrate a source for the bleeding (Figure 3). There was fluid in the upper part of the haemorrhage and its preretinal location was confirmed by fluorescein angiography. There was neither posterior vitreous detachment nor a hole in the posterior hyaloid.

The patient was discharged home on day 10. After 15 days, the subhyaloid haemorrhage had completely cleared. The intragel haemorrhage cleared after 2 months with no further changes. The visual acuity was sustained at 20/20 during a follow-up period of 4 months. No retinal damage or rebleeding occurred due to the laser treatment, and vitrectomy was not required.

Figure 1. **Fundus photograph of the right eye showing a round, well circumscribed, dome-shaped, dark premacular subhyaloid haemorrhage centred at the fovea. The fluid level in the upper part of the haemorrhage suggests subhyaloid space location.**



Discussion

Subhyaloid haemorrhage in the premacular space may cause an acute, dramatic loss of central vision, which may persist if left untreated. Subhyaloid or subILM haemorrhage is usually caused by diabetic retinopathy,[1] hypertensive retinopathy,[2] retinal artery macroaneurysm,[2] Valsalva retinopathy,[3] Terson syndrome,[2] blood dyscrasias,[4] bacterial meningitis,[5] vitreoretinal traction of different origins, or trauma, or occurs spontaneously following partial detachment of the posterior hyaloid membrane, central retinal vein occlusion, blunt ocular trauma, laser in situ keratomileusis, macroaneurysms, presumed ocular histoplasmosis syndrome, idiopathic central serous choroidopathy, choroidal rupture, or age-related macular degeneration.

Subhyaloid haemorrhage may either improve spontaneously or may require therapeutic intervention to prevent secondary retinal degeneration. Different therapeutic approaches have been adopted for treatment of this condition, ranging from conservative treatment to prompt vitrectomy. Sung et al recommended that premacular subhyaloid or subILM haemorrhage caused by factors other than diabetic retinopathy may be conservatively managed for the first few months.[6] However, since subhyaloid haemorrhage may be associated with permanent macular changes before it spontaneously resolves and adequate treatment of the underlying cause of the haemorrhage may be delayed with potential risks for further damage to ocular structures, early intervention seems to be crucial.

O'Hanley and Canny showed that patients who did not receive vitrectomy within 4 weeks of the onset of the haemorrhage progressed to late macular traction and visual acuity no better than 6/30.[1] These authors showed that possible toxic damage to the retina occurred due to prolonged contact with haemoglobin and iron. Certainly, visual function can be almost instantly restored by pars plana vitrectomy with surgical separation of the posterior hyaloid membrane and evacuation of all blood. However, vitrectomy, although a routine procedure, is associated with numerous side effects.[7] The progression of lens nuclear sclerosis, even after uneventful vitrectomy is a well known complication, which occurs in almost all patients.[7] Intraoperative retinal breaks and postoperative proliferative vitreoretinopathy may result in retinal detachment and severe loss of visual function.[7] Tissue plasminogen activator (tPA) and perfluoropropane (C_3F_8) injection are alternative ways to clear a subhyaloid

Figure 2. **The haemorrhage instantaneously drained into the vitreous cavity at Nd:YAG laser hyaloidotomy.**

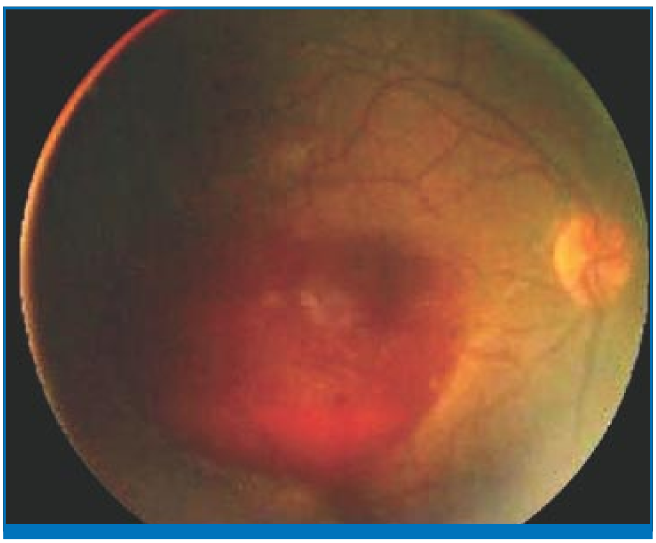


Figure 3. **Fluorescein fundus angiography at day 4 after the hyaloidotomy showing the clearance of the haemorrhage with the residual inferior rim of haemorrhage outlining the detached internal limiting membrane.**

haemorrhage, especially when the patient has media opacity or when there is a problem with contact lens application for laser therapy.[8,9] Furthermore, it has been reported that intravitreal tPA and sulphur hexafluoride (SF_6) may induce pneumatic displacement of premacular subhyaloid haemorrhages in shaken and battered baby syndrome.[10]

Nd:YAG laser photodisruption of the posterior hyaloid membrane has been reported to achieve distribution of the haemorrhage in the vitreous, which resulted in accelerated clearing and visual improvement.[11] Within the first 3 to 4 days after occurrence of a premacular subhyaloid haemorrhage, the posterior vitreous boundary layer may be lacerated by argon laser coagulation in such a way that the blood floats into the vitreous body, where it is absorbed within a few weeks. Older premacular haemorrhages under an intact vitreous boundary layer, typically green-white in colour, should be treated by more invasive vitreo-surgical procedures.[12] Tassignon et al recommended Nd:YAG laser for treatment of premacular haemorrhages without underlying proliferative disease and where the volume of the blood does not greatly exceed 12 µL.[13] Spontaneous resorption of subhyaloid or subILM haemorrhage caused by Valsalva retinopathy usually occurs without sequelae.[3] Berrocal et al showed that patients without subretinal neovascular membranes had a better visual improvement rate.[14] YAG laser was previously reported to be useful for lysis of organised vitreous membranes near the optical axis of the eye.[15] The advantages of Nd:YAG laser over vitrectomy are the ambulatory and painless nature of the procedure, without stimulating proliferative vitroretinopathy. In addition, the use of Nd:YAG laser will not inadvertently affect the outcome of a later vitrectomy.[16] Complications included macular holes and retinal detachment from a retinal break in a patient with myopia.[17] For patients with cataract or media opacity, effective and precise laser delivery could be difficult.[11]

This case demonstrates the effective treatment of a dense central subhyaloidal haemorrhage using Nd:YAG laser. No side effects were attributed to the procedure, in contrast to the potential risks of vitrectomy. Since the haemorrhage may cause permanent macular changes before it resolves, Nd:YAG laser hyaloidotomy, being a safe and effective procedure, achieving rapid resolution of premacular subhyaloid haemorrhage with restoration of binocular vision and preventing the need for vitreoretinal surgery, is a viable treatment alternative for eyes with recent bleeding in selected patients. The risks and benefits of Nd:YAG laser treatment as a routine procedure should be evaluated in a randomised trial and compared with those of deferral of treatment or primary vitrectomy.

References

1. O'Hanley GP, Canny CLB. Diabetic dense premacular haemorrhage. Ophthalmology 1985;4:507-511.
2. Gass JDM. Stereoscopic atlas of macular diseases: diagnosis and treatment. 3rd ed. St Louis: Mosby; 1987:361-367;560-564.
3. Duane DT. Retinopathy and distant extraocular trauma. Duane's clinical ophthalmology. Vol 3. Philadelphia: Lippincott; 1989;32:3-4.
4. Cunningham RD. Retinopathy of blood dyscrasias. Duane's clinical ophthalmology. Vol 3. Philadelphia: Lippincott; 1989;18:1-8.
5. Fraser SG, Horgan SE, Bardavio J. Retinal haemorrhage in meningitis. Eye 1995;9:659-660.
6. Sung VC, Murray DC, Price NJ. Subhyaloid or subinternal limiting membrane haemorrhage in meningococcal meningitis. Br J Ophthalmol 2000;84: 1206-1207.
7. Schmitz K, Kreutzer B, Hitzer S, Behrens-Bauman W. Therapy of subhyaloidal haemorrhage by intravitreal application of rtPA and SF_6 gas. Br J Ophthalmol 2000;84:1324-1325.
8. Adel B, Israel A, Friedman Z. Dense subhyaloid hemorrhage or subinternal limiting membrane hemorrhage in the macula treated by Nd:YAG laser. Arch Ophthalmol 1998;116:1542-1543.
9. Iijima H, Satoh S, Tsukahara S. Nd:YAG laser photodisruption for preretinal hemorrhage due to retinal macroaneurysm. Retina 1998;18:430-434.
10. Conway MD, Peyman GA, Recasens M. Intravitreal tPA and SF_6 promote clearing of premacular subhyaloid hemorrhages in shaken and battered baby syndrome. Ophthalmic Surg Lasers 1999;30:435-441.
11. Gabel VP, Birngruber R, Gunther-Koszka H, et al. Nd:YAG laser photodisruption of hemorrhagic detachment of the internal limiting membrane. Am J Ophthalmol 1989;107:33-37.
12. Kroll P, Busse H. Therapy of preretinal macular hemorrhages. Klin Monatsbl Augenheilkd 1986;188:610-612.
13. Tassignon MJ, Stempels N, Van Mulders L. Retrohyaloid premacular hemorrhage treated by Q-switched Nd-YAG laser. A case report. Graefes Arch Clin Exp Ophthalmol 1989;227:440-442.
14. Berrocal MH, Lewis ML, Flynn HW Jr. Variations in the clinical course of submacular hemorrhage. Am J Ophthalmol 1996;122:486-493.
15. Chung J, Kim MH, Chung SM, Chang KY. The effect of tissue plasminogen activator on premacular hemorrhage. Ophthalmic Surg Lasers 2001;32:7-12.
16. Veken AVD, Velde FVD, Smeets B, Tassignon MJ. Nd:YAG laser posterior hyaloidotomy for the treatment of a premacular vitreous floater. Bull Soc Belge Ophthalmol 1997;265:39-43.
17. Ulbig MW, Mangouritsas G, Rothbacher HH, Hamilton AM, McHugh JD. Long-term results after drainage of premacular subhyaloid hemorrhage into the vitreous with a pulsed Nd:YAG Laser. Arch Ophthalmol 1998;116:1465-1469.

Address for Correspondence

Dr Yusuf Akar
Akdeniz Üniversitesi Tıp Fak.
Göz Hastalıkları ABD
07070, Antalya, Türkiye.
Tel: (90 242) 228 5050
Fax: (90 312) 227 4490
E-mail: dryakar@yahoo.com